A routine phenolsulphonephthalein function test before any treatment is given will sometimes reveal seriously impaired kidneys and warn against the administration of harmful amounts of arsenic or mercury.

I was very much interested in Miller's remarks concerning the predisposing influence of seborrheic tendencies to post-arsphenamine eruptions.

I do not agree with Miller that it is desirable to wait for a week or more following the appearance of a post-arsphenamine eruption before administering sodium thiosulphate. If this drug is harmless, as everyone seems to agree, and if it acts as an antidote to a poison, as experience has apparently demonstrated, then, theoretically, the sooner it is given, the less will be the toxic effects from the arsenic. The skin is not the only tissue which may be affected. I have seen a case of acute yellow atrophy of the liver come to autopsy following the administration of neoarsphenamine.

Albert M. Meads, M. D., Hutchinson Bldg., Oakland—Miller has emphasized the importance of watching for minor skin eruptions, as well as for severe eruptions, following the injection of any of the arsphenamines. The relationship between such eruptions and the amount of salvarsan given at each injection, in our experience, has been noticeable. The syphilitic clinic of Alameda County Hospital has made it a point not to give more than six decigrams of neosalvarsan at a single dose in routine treatment, and we have found that severe reactions of all kinds have not been evident. No severe eruptions have been noticed and but two minor eruptions seen in women following the first injection. Subsequent injections did not aggravate the condition, so there is a question as to the origin of the rash. Clinic cases, of course, are not watched as carefully as they might be, the patient being trusted to report any unexpected occurrence. A severe eruption at least would have been reported to the physician administering the drug, but this has not been the case. About fifty cases a week, men and women, are under treatment at the present time.

In my private work my experience is limited to one case of exfoliative dermatitis occurring after the second dose of neosalvarsan in a non-luetic case, and to a few cases of transient oedema of the lips, face, and other parts, occurring almost immediately after the injection and clearing up quickly under a small dose of adrenalin. Possibly I have overlooked the less pronounced eruptions, but I can say that since adopting less than the maximum dose there has been a decided decrease in reactions in any form in cases under my observation. Certainly Miller's paper will cause me to be more watchful for the less pronounced eruptions and will prompt me to be a little more cautious in pushing arsphenamine in such cases.

Hiram E. Miller (Closing)—I had hoped that in the discussion someone would give us his experience in treating post-arsphenamine eruptions with sodium hyposulphite or the relative number of cases he had seen develop after arsphenamine and neoarsphenamine therapy. Ayers mentions the value of phenolsulphonephthalein function tests. They are of definite value in determining renal function but the post-arsphenamine eruptions are apparently due to a hypersusceptibility to arsenic and lowered renal function plays a minor part. In the eighteen cases in my series, the urine findings were normal in all except two cases, and in these two only for two days during the beginning of the eruption was there any albumen present in the urine.

We should aim to take every precaution to prevent these eruptions and once they have occurred to use every means at our command in clearing them up. Sodium thiosulphate intravenously and orally is of definite value in the treatment of the eruption.

THE MANAGEMENT OF FRACTURE OF THE BASE OF THE SKULL*

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Fracture of the base of the skull per se has no mortality. Death is due to intracranial injury and not to displacement of the fragments. In the severe crushing injuries with gross bony changes, the accompanying cerebral traumatism is so severe that death occurs immediately or within the first few hours.

In the cases that survive this period, the accompanying hemorrhage or secondary edema is responsible for death from the increased intracranial pressure.

A review of the vital statistics of the District of Columbia for the past twenty years shows a steadily rising death rate from fracture of the skull. Though the total death rate from all causes has fallen off from 21.74 per 1000 in 1900 to 14.77 per 1000 in 1920, the death rate from external and violent means has risen from .74 per 1000 in 1900 to .83 per 1000 in 1920. An analysis of the causes of violent death reveals again a steadily increasing percentage due to fracture of the skull.

In 1900 11 per cent of the violent deaths were caused by fracture of the skull.

In 1910 14.8 per cent, and in 1920 15.9 per cent.

As might be expected, 66 per cent of deaths from fracture of the skull occur in the first four hours after injury. Twenty-four per cent within seventy-two hours to ninety-six hours, and 10 per cent after that time. It is probable that the larger group of immediate or very early deaths cannot be affected by treatment, but there is left an appreciable percentage of cases (33 per cent) that should be improved by a better understanding of the problems involved.

As meningitis was only mentioned as the cause of death in two of all the cases of fracture of the skull, we can assume that in the vast majority this is a negligible factor.

Fractures involving the base of the skull are linear in type. There is no bony deformity, but from the anatomical conditions, they are always accompanied by injury to the basilar dura and intradural hemorrhage. As is well known, the dura over the vault is loosely attached to the bone and usually not injured in fractures in this region, unless there is a depression with indriven fragments. When hemorrhage occurs in fractures of the vault it is produced by tearing of the meningeal arteries that groove or tunnel the bone. In such event the hemorrhage is extradural. The hemorrhage is limited to the immediate injury, and as it advances it dissects the dura from the inner table. In these cases, the accumulation of pressure is gradual, easily associated with the "lucid interval" and manifests itself by localized pressure with mono- or hemiplegia.

The dura over the base of the skull is so intimately attached to the bone that even a slight fracture is always accompanied by injury to the dura and the escape of the resulting hemorrhage into the

* Read before the Utah State Medical Association, June 20, 1923.

subdural or subarachnoid spaces. This hemorrhage may be arterial or, when the dural sinuses are injured, venous. The hemorrhage meets with little or no resistance, and bleeding occurs freely between the dura and the brain. The result is a relatively rapid increase in intracranial pressure.

Most fractures of the base of the skull are compound, in that the paranasal sinuses or the middle ear is involved. This makes itself manifest by hemorrhage from the nose, mouth or external auditory meatus. Linear fractures of the posterior fossa not involving the temporal bone are usually not compound. The communication of the fracture, even indirectly with the outside, of course adds to the possibility of infection as a dangerous complication. It is, however, surprising how seldom death occurs from this factor, and as I have said above, there are only two cases in the statistical study where meningitis was given as a cause of death in the fractures of the skull.

The clinical picture of fracture of the base of the skull is characteristic. The immediate loss of consciousness accompanying the injury due to concussion is followed by a varying degree of mental disturbance that depends on the cerebral injury and the resulting compression. In the early hours, the blanched skin, the clammy sweat, the dilated pupils, and the rapid pulse of the unconscious patient bespeak the immediate contusion of the brain. If compression supervenes from hemorrhage the unconsciousness will continue, but the clinical picture will gradually change. The pulse becomes slow and full, the face and scalp congested, the breath stertorous and labored, and the flaccidity will give way to restlessness and semi-consciousness that make the patient extremely hard to control. The blood pressure in the early stage of concussion is low, but with the onset of compression, the systolic pressure will rise, while the diastolic pressure tends to lag behind. As pressure increases unconsciousness depends, and the restlessness gives way to deepening coma.

The mechanism underlying these phenomena is well known. With the addition of free hemorrhage within the non-expansible cranial cavity, first the cerebral fluid is displaced, then the venous blood from the thin-walled veins and finally, if the pressure continues to increase, the arteries themselves are obstructed, with the resulting anemia of the brain. In an effort to maintain the cerebral circulation against the increasing intracranial pressure within the skull, the systolic pressure is gradually increased and the cardiac impulse slowed. It is during this period of gradually increasing intracranial pressure that well-directed treatment may save life. If the pressure is allowed to advance to the point where the intracranial pressure rises above the systolic pressure, Kocher's fourth stage of compression develops, and the time for interference has passed.

In the management of cases of fracture of the base of the skull, nothing can be done in the group of severe crushing injuries with immediate death, or death within the first few hours. It is the gross cerebral injuries that cannot be controlled and cannot be repaired that are responsible for the mortality. In the cases that survive this immediate mortality, control of the complications will definitely reduce the number of deaths. Our aim should be to ascertain as early as possible the degree of intracranial pressure. If it is high and the patient already in the second or third stage of compression, the cerebral circulation is threatened and indications are definite for immediate interference. The situation is a desperate one, and heroic measures may be necessary to control the rapidly increasing intracranial pressure. Here Cushing's decompression operation is useful. Such an operative procedure, however, should add not one ounce more to the load the organism is laboring under. For that reason a general anesthetic, such as ether or even gas-oxygen is definitely contraindicated. Frequently no anesthetic at all will be necessary, but even with a comatose patient any attempt at manipulation or operation will arouse him to a restlessness that will make surgical procedure impossible. The indications are definite for local anesthesia in decompression of the fracture of the base of the skull. The operative area should be infiltrated with one per cent novocain, preceded by a heavy hypodermic of morphine if necessary. A right-sided subtemporal decompression is the operation of choice, though the evidence of maximum pressure elsewhere may justify decompression in that region. The Cushing operation, through a vertical incision and separation of the fibers of the temporal muscle, exposes the skull where the bone is thinnest and most easily removed. A stellate incision of the dura allows the escape of blood and bloody cerebrospinal fluid and decompresses the silent temporal lobe. The resulting brain hernia compensates for the increase of pressure from the accumulated hemorrhage. If a unilateral decompression does not suffice, the procedure should be immediately repeated on the opposite side. If hemorrhage is free, drainage may be necessary. In certain cases a torn sinus, if accessible, can be occluded by a muscle-graft.

In the less desperate type of case, where indications do not point to an extreme pressure, but where the diagnosis of a fracture of the base of the skull is clean-cut, a question of judgment will often decide the life or death of the patient. In such cases we have many guides as to the progress of the intracranial pressure. The old clinical signs of slowing pulse and deepening coma may not be sufficient to tell us definitely enough of what is going on within the skull. Careful and frequent blood pressure readings made by the same observer are of great value. Ophthalmoscopic examination in the hands of an expert may show a gradual advance of pressure in the retina. Again, a carefully performed spinal puncture with or without the use of a manometer will contribute materially to our knowledge of the case. If any of these signs or symptoms indicate a definite and steady rise of intracranial pressure, then our aim should be to control that rise as best we may.

When the signs and symptoms indicate a mild and gradual increase of intracranial pressure, use of hypertonic salt solution may control the situation. The intravenous injection of 100 to 200 cc. of hypertonic salt solution will temporarily reduce brain-bulk and decrease the intracranial pressure.

This effect, however, is temporary and if used will necessarily have to be repeated. Since hypertonic salt solution injected into the intestine has the same effect experimentally, Dowman of Atlanta has suggested the frequent and repeated administration of full doses of magnesium sulphate by mouth. In the treatment of this class of cases he has found a distinct benefit from its use, and, with a very limited experience with it, I can indorse his claim.

When there is a more rapid and more distinct increase in intracranial pressure following the injury in a case of fracture of the base of the skull and where one hesitates to operate, spinal puncture drainage will often suffice. It should be used, however, with extreme caution. If the pressure is at all high and where doubt exists as to the safety, it is probably wise to discard this treatment for decompression under local anesthesia. If, however, spinal puncture is used, it will probably have to be repeated.

Careful observations should be frequently made. If the treatment outlined above does not control the advancing increased intracranial pressure, then operative interference should be resorted to immediately.

In cases of fracture of the base of the skull where immediate compression has been controlled so as not to become excessive, we occasionally see a recurrence of compression several days after injury. In these cases the compression is caused by secondary edema that follows the original cerebral contusion. In these cases the use of hypertonic salt solution or repeated doses of salines may be most effectual. However, as before, if their use does not control the pressure, one should not hesitate to perform a decompression.

The value of decompression in fracture of the base of the skull is still a debatable question. There have been statistics published both for and against its use. Mixster of Boston analyzed the cases for a period of twenty-seven years in the Massachusetts General Hospital. Of the cases treated up to 1900, 9 per cent were operated upon with a total mortality of the entire series of 68 per cent. Of the cases treated between 1900 and 1911, 23 per cent were operated upon with a mortality of 54 per cent. Of the cases treated between 1911 to 1917, 71 per cent were operated on with a mortality of 36.5 per cent. These figures strikingly demonstrate the decrease of mortality where operation was used. His figures again show the value of operation in the early stages of compression rather than operation as a desperate last resort. Of the operated cases where operation was performed within the first forty-eight hours, the mortality was only 41.2 per cent, while in those so treated more than forty-eight hours after injury the mortality was 71 per cent. In a similar study of a small series of cases at the Garfield Memorial Hospital I find that the mortality in cases where operation was not used was just about twice that of the mortality in cases where operation was made part of the treatment. My experience, therefore, convinces me that decompression is definitely indicated in all cases of fracture of the base of the skull that show increasing intracranial pressure. This decompression may be accompanied by the use of hypertonic salt solution or salines by mouth, by repeated lumbar puncture or finally by subtemporal decompression operation.

CONCLUSION

1. With the exception of the group of cases with severe crushing head injury and death within the first few hours, the mortality in fractures of the base of the skull is almost entirely due to increase of intracranial pressure from early hemorrhage or late edema.

2. Careful observation and repeated notations of the blood pressure, pulse rate, retinal changes and spinal fluid pressure should be made in all cases.

3. A slightly increasing pressure may be controlled by intravenous injection of hypertonic salt solution or the administration of saline by mouth.

4. In the cases with rapidly rising pressure or where the above does not suffice, decompressive operation should be performed under local anesthesia.

6. The use of decompression either by hypertonic salt solution, spinal puncture or operation will definitely decrease the mortality in the cases under consideration.

Reciprocal Responsibility of Health Officer and Other Public Officials—The activities of any public service board should be free from political influence. The ideal condition, so far as such is obtainable, would be to have the service divorced entirely from all interference on the part of those persons holding positions by virtue of their political affiliations. A health board or any other public board, hampered by political machinations, is a travesty on efficiency. Discord and lack of harmony among the members and employes is the immediate and direct result. Aside from internal disruption, which is a corollary of outside interference, there is the more important result of the loss of usefulness in the service to the public. Therefore, to insure a maximum of efficiency, the appointments should be made solely on a basis of merit and fitness, and the term of office determined by results. . . .

Volunteer health organizations have their responsibility. With a reciprocal attitude, they are valuable adjuncts to the activities of the health officials, and, if the program is suggested by the health authorities, they may be eminently useful. But there must exist a mutual appreciation of the functions and prerogatives of both, and an acknowledgment by these auxiliary bodies of the right of domination in health activities by the State board. . . .

Scientific facts on which health work is founded are as certain as those which lie at the basis of astronomy, for example. Both are the practical development and application of fundamental scientific principles and, however divergent in most respects, the physical laws which are operative in one are wholly applicable to the other. (Oscar Dowling, Journal A. M. A., July, 1923.)

Discussion (by C. W. Garrison, Little Rock, Ark.)—Public health is challenging the health officer of today to initiate a program of right living, involving both eugenics and environment. Public health is as distinctly a branch of medicine as is surgery or obstetrics, and a health officer should not only be as highly trained technically in his specialty as the surgeon, but should possess unquestionable honesty, adaptability, courage, leadership, and diplomacy.